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APHASIA.[1]

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The problem of aphasia, a few years ago, has been treated monographically by a number of writers, such as Wyllie, Bastian, Bramwell and Elder, and, in this country, Langdon and Collins, by Miraillié, Pitres, and Bernard, in France, and the treatises of Mills, v. Monakow, and Dejerine added much of importance to the standard descriptions of Kussmaul, Lichtheim, Ross, Gowers and the pupils of the Salpêtrière. Aphasia has since passed into one of those stages of self-sufficiency which are so apt to retard progress because of excessive faith in the theoretical constructions and the idea that far more is solved and proved than is really the case. Throughout the literature on aphasia certain 'elements' of psycho-physical correlation are taken for granted, often enough without much concern as to the strength of their foundation, merely for plausibility's sake. The appearance of a review of the field by Wernicke furnishes some material concerning the problem of elements supported by the available data of aphasia, because Wernicke is a decided localizer, and yet strongly enough opposed to reading and writing centers to subject them to an extremely laborious and searching discussion. Wernicke gives in this 'lecture' very interesting and clean-cut statements of points which should be within reach of every worker in this rather neglected field; and also psychologically instructive illustrations of his method of combination of analysis and reconstruction, and, with it all, he rouses a new hunger for further casuistic evidence and for greater clearness concerning the concepts with which one works, or which one has reason to think are aroused in most readers.

[1] Wernicke, C., *Der aphasische Symptomencomplex.* 13th lecture of Die Deutsche Klinik (Berlin and Wien, Urban & Schwarzenberg, 1903), Vol. VI., pp. 487–556.

[2] This number has been prepared under the editorial care of Dr. Meyer.

The central issue of Wernicke's lecture is the relation of spoken and written language and the bearing of the 'word-concept' or 'word-notion.' He begins with a brief statement of a case of so-called pure or isolated agraphia (reported in full, *Monatssch. f. Psych. & Neurol.*, April, 1903). The patient is a woman forty-six years old; the symptoms had developed within nine months, first slowly with increasing weakness of the right hand, then with three more acute exacerbations; the second one brought a transitory loss of speech, leaving slight anarthria, and the third one a permanent picture of right hemiplegia and profound sensory disorders of the entire right side, and complete loss of spontaneous writing; only once the patient had been able to draw, under dictation and special urging, the letter 'a' and the figures 2, 3 and 4. Understanding and speech were perfectly normal, even the reading and understanding of letters and figures and words and of outlines and pictures. Any attempt to write —with chalk and blackboard, *i. e.*, with such movements as the right hand had not lost in the hemiplegia — led to a striking perplexity and expression of exhaustion.

It would seem very tempting to assume that such a condition of 'pure agraphia' would be referable to the incapacity of a special writing center, a loss of the memory of how to write words, just as a lesion of Broca's center is supposed (also by Wernicke) to lead to a loss of the memory of the movements necessary for speech. Wernicke gives, however, good reasons why the accepted views about a speech-utterance center should not be generalized, and that the assumption of a writing center would be premature, if not really erroneous. He predicts a lesion largely of association-paths (the centrum ovale underneath the posterior angle of the island).

Wernicke's historical sketch illustrates splendidly individual differences in the psychology and methodology of investigation. Meynert is given a very prominent position. His teachings are said to have given the clinical and experimental data of Broca and Hitzig the real foundation and to have furnished Wernicke the material for the assumption of a sensory speech center in the first temporal gyrus. 'A happy coincidence soon corroborated this supposition by two autiosies' — an excellent illustration of how constructive imagination sharpens the attention needed for discovery. The discrepancy between Broca and Trousseau became intelligible, and the data seemed sufficient to attempt, with the help of diagrams, a synthetic reconstruction of the functions decomposed by the experiment of nature.

Just what should be assumed as safely established elements for

such reconstructions was probably considered too easy a matter at first. Wernicke thought of explaining all the facts out of the function of two centers and their connection, out of some data from the method of learning a foreign language, and the acquisition of language by the child — unfortunately a field of speculation rather than of safe knowledge even to-day, — and out of the rough clinical and localizatory experiences in aphasia. The sensory word center is the place where 'sound-images' have their nerve-cells or cortical elements. He thinks that the sound-appreciation is the first acquisition, to be followed by the acquisition of appreciation of its sense, or 'secondary identification.' What happens in learning a foreign language supports this differentiation. In connection with the word-sound concept, the child acquires a word-utterance concept by manifold practice; and the firm connection of the two is identified with the possession of the word-concept or *word-notion*, or what the French call 'internal language.' (Special decomposition of the words into letters is considered a secondary process.) The acquisition of the word-notion or word-meaning is the most important process in learning a language, and, for correct use of language, the integrity of 'both the sensory and the motor component' of the word-meaning would seem indispensable. Wernicke does not, however, follow Bastian in assuming that all speech function is a recapitulation of how words are acquired (a view which makes the most of the supremacy of the word-hearing center); he claims that after destruction of the sensory center articulated language is preserved; the speech-impulses from the entire remaining cortex reach the 'word-notions' directly and, where the latter are mutilated, the speech-movement images, so that articulated language remains, though defective owing to loss of the regulating influence. These data 'should be sufficient to understand the clinical picture of sensory aphasia; the chief symptoms can readily be derived from them.'

The scrupulous reader could hardly share such a faith in the safety and definition of the 'elements' offered. For 'sound-images' he is referred to the 'cortical elements of the sensory speech-field' without an inkling as to how they would work. The word-notion is said to have its substratum in the connection of definite elements of the sensory center with the corresponding definite elements of the motor center — a far-reaching claim, considering that there is not, as yet, any evidence of subdivision of the 'center.' Something of a substratum with a 'word-notion' is admitted to persist outside of this complex of direct connections, because destruction of the sensory center

does not necessarily abolish articulated speech or utterance. Wernicke is not explicit as to the make-up and localization of the substratum and nature of the 'word-notion.' He merely says: "Such a firm connection of memory-images which belong together constitutes the essence of 'Begriff' (of the idea, concept or notion)." The question is whether that which lies outside of the speech-field should or should not have a definite share in the 'word-notion.' This is, undoubtedly, a crucial point for any attempt at explaining language in terms of activity of special cortical *elements* of clearly limited speech-centers. Considering the revival of difficulties of histological definition of 'nerve-elements,' and the logical or verbal rather than functional abstraction of 'elements' in speech function, it seems hazardous to promise the possibility of deduction of the picture of sensory aphasia from the few elementary conceptions given. It should certainly be clearly understood that, so far, we can only contrast very broadly the apperceptive and the emissive functions as Ross called them; and that a discrimination of the actual 'elements' and the concept 'word-notion' is a merely temporary contrivance.

Wernicke's characterization of the clinical types is lucid and definite, and rendered here for comparison with the claims of other writers. He begins with *sensory aphasia:* Although there is no deafness or not enough to account for the disorder, the patient presents a defect of understanding of the word-sound and of the word-sense, as far as the patient depends on the interpretation of sound, while gesture and non-auditory signs are easily grasped (and must be guarded against in tests!). Articulated speech is preserved and even excessive, 'perhaps owing to the numerous misunderstandings.' "For although the patient uses a fairly rich vocabulary and good form of speech, he frequently blunders in the choice of words, and even uses wrong or disfigured words without being aware of it; under affect he usually speaks better."

Objects shown are usually wrongly named, often with paraphasia. The confusion of words in spontaneous speech may reach the degree of true unintelligible jargon-aphasia. What the patient replies does not start from words heard; loss of understanding of the word sound necessarily also frustrates repetition. Written language, depending on the word-concept or internal language, is always strongly affected in sensory aphasia. It is, however, not well studied, since it is not common property of all persons. *The onset* is usually acute, through occlusion of a vessel, usually with very slight shock and often without any hemiplegia. As to prognosis, Dejerine thinks it to be a lasting

defect; Wernicke, with most writers, accepts *restitution by reëducation* from ordinary life; difficulty in the understanding of rare words — he mentions vertebral column, knee-pit, arm-pit — and also in the *naming of objects* is the most persistent residual. The restitution of *written* language is not sufficiently studied to allow of generalization.

With a little consideration one is struck by the haziness of the elements, loss of which should account for the variations in the extent of individual clinical differences, the varying affections of the word material or word-concepts, and the individual differences in restitution, and such matters as the difficulty of naming objects after recovery of spontaneous speech. It is also striking how sadly deficient the literature is in such a simple matter as a good description of the accurate extent of lesions in terms which should stand the critical attitude of a Flechsig. With all the observations collected by Miraillié and Bastian (Wernicke does not dispose of all the cases opposed to his views), we still are in a very vague position, far from being able to deduce the concrete symptom-complex from a simple scheme.

Motor aphasia or aphemia is 'equally easy to describe.' (1) Articulated language or the mechanism of articulation is 'forgotten'; hence there is mutism with but few residuals, often only of senseless syllables or words, and even these are not used at will but automatically (recurrent utterances). In emotion or in sleep, words not otherwise available may be produced. There is no bulbar palsy, but frequently a slight hemiplegia, or slight hypoglossal palsy, not sufficient to explain the aphemia; for some time the patient may be unable to show the tongue, to puff the cheeks, gnash the teeth or even open the mouth to order without sticking out the tongue; these disorders are, like the aphemia, a defect of the memory of the way to do things, and often exist only in the first period of the aphemia. (2) The *understanding* of speech is largely correct; orders are correctly carried out, and mimic well responded to; but Wernicke admits now that there is *at least some difficulty for longer sentences* as Dejerine has shown; but this difficulty is usually open to improvement. This disorder of understanding is explained by the fact that the acquisition of speech-concepts is a fundamental phase in the learning of a language, and that their loss, in turn, has a variable influence on language as a whole. In this respect there are evidently individual variations (not further specified). (3) *Writing* keeps pace with articulated speech. *The onset* of motor aphasia is usually with a more marked shock and more or less right-sided hemiplegia. The *prognosis* is on the whole unfavorable except where the insult is merely slight or the interfer-

ence merely indirect (due to a lesion of a neighboring part, or not very infrequently a very severe insult of even the *right* hemisphere with left-sided hemiplegia). Restitution usually leaves much exaggeration of motion and slowness; a certain awkwardness and exaggerated effort resembling that of deaf-mutes who have learned to speak, and syllabic stumbling always remain even in favorable cases. *Repetition* remains as deficient as spontaneous speech. *Training* by optic methods, as in deaf-mutes, seems to give very favorable results (in six weeks — Dejerine and Thomas). In relatively rare cases the motor defect is not so complete. The patient may succeed in repeating easy words, or short sentences, but *never* more complicated words or sentences; vowels or syllables without any resemblance will be substituted, or the patient's own name, or an 'ach Gott.' *Partial motor aphasia does not seem to exist* beyond these exceptions.

This presentation gives a much more exclusive definition of the functional picture and its clinical evolution than is suggested by most English and American writers, who describe several types of cortical motor aphasia, and in turn, are more hopeful about the anatomical focal subdivision of the motor speech-field into a propositionizing and utterance center. One of Wernicke's claims, especially worth reiterating and testing, is the non-existence of partial motor aphasia. The function of the 'center' evidently is considered one 'en bloc,' not a sum of many individual word utterance memories.

Wernicke still inserts here his hypothetical *conduction aphasia.* The available empirical data are admitted to be scanty and not consistent. Paraphasia is not sufficient evidence of the interruption of the connection between hearing and utterance center. It can result from more causes than Wernicke first assumed. Nor does repetition of words heard prove the integrity of this simple path. The sound-picture seems to be sufficient for an understanding at least of ordinary words (Wernicke explains the recovery from sensory aphasia on this ground, *i. e.*, without the creation of a new auditory word-center!), and motor utterances may be roused spontaneously without a previous rousing of the sound-picture; therefore, destruction of the connective path will not necessarily frustrate the repetition of words to order, as long as they are *understood.* *Some* paraphasia will then occur, realized by the patient. Evidence of the *integrity* of the oldest and original conductive path would be furnished by automatic echolalia and by *repetition of foreign or senseless words.* Wernicke, therefore, looks for a case in which *merely* echolalic repetition would be *destroyed*, with preserved understanding and execution of speech, and

a paraphasia with realization of the mistakes. The recorded cases of lesions of the island demand such a restriction of the theoretical deduction if they are not directly opposed to the whole conception. And what becomes of the simplicity of the notion 'word-concept'? In conduction-aphasia it should be destroyed; but Wernicke explicitly admits the existence of a long-circuit substitute.

Convinced of the anatomical and clinical demonstration of a sensory and a motor speech-center, Wernicke proceeds to the construction of pure or subcortical aphasias as 'a necessary logical consequence.' There must occur cases in which the projection-system of the one center or the other is destroyed without interference of the centers themselves and their connection; these cases too show a loss of understanding of the word-sound, or a loss of articulated speech, but 'preservation of the internal language or intact word-notions.'

In *pure aphemia* (not infrequent, especially with hemiplegia), Wernicke finds some evidence of the correct intention of utterance, and the attempt at repetition is never so completely miscarried as with cortical destruction. Understanding is intact even for complicated sentences. *Written language* is quite *intact*. Dejerine published the first convincing cases with a lesion just beneath Broca's convolution, above the internal capsule, and demonstrated on them the fallacy of Charcot's view of a special writing center. Integrity of the word-concepts becomes the formula for the fact that the patient can write. In cases of illiteracy, Lichtheim has suggested the test of counting the syllables to demonstrate the integrity of the word-concept. The best sign, according to Wernicke, is the correct intonation of the speech rests, which should be in harmony with the rhythm of the intended utterances. Since the intonation-test necessarily fails where the patient has no speech rests, on which to produce the intonation, or where he does not grasp the issue of the test, one would welcome the simple contrivance of Onuf and Fraenkel, who merely depend on simple counting (turning over every sixth card of a pack) as evidence of integrity of internal language. I have, however, just recently had a chance to examine a patient of Doctor P. L. Murphy of Morganton, N. C., with motor aphasia in a state of partial reconstruction, who succeeded at once with the card test, *although* he has not recovered his writing as well as his speech, and gives no introspective evidence of knowing the words which he is not able to produce. This simple test can, therefore, no longer be accepted as sufficient evidence of what it claims to demonstrate clinically, not to speak of the danger of using it for anatomical inferences. The number of clinically and anatomi-

cally well established cases of subpictorial apnasia with really purely subcortical lesion is actually very small, and hardly larger than the number of cases in which the same clinical symptoms coexisted with destruction of the cortex itself. This is a serious objection to Wernicke's categorical attitude. It should be understood that the *collection and publication of such cases with all the clinical and anatomical detail is still urgently to be desired.*

Pure word deafness implies simple loss of understanding of words notwithstanding sufficient hearing, with integrity of word-concepts and all modalities of speech. Of this disorder Wernicke admits only one case of Liepmann as clinically and anatomically beyond doubt.[1]

Wernicke next passes to a *plea for the transcortical aphasias* due to interruption of the 'connection of the motor or the sensory word centers with the concept regions.' Such a center for the 'word sense' or concrete concepts is here explained as a mere fiction, representing the firm connection of the visual, olfactory, auditory and tactile memories, which necessarily are complete inter- or transcortical complexes. (Wernicke declines to accept Flechsig's association or coagitation centers; all these fields have projection systems; 'the island and its claustrum might alone pass as association organ,' in faithful allegiance to Meynert!) *Transcortical sensory aphasia* is a loss of the word-sense with preserved appreciation of the word-sound — the active component of audition, repetition, is preserved (with but moderate para-

[1] The other spurious cases are made the basis of a discussion of some interest. Another of Wernicke's pupils, Freund, had tried to trace pure word deafness to a peripheral affection (of the labyrinth); this led to the utilization of Bezold's statement concerning the necessary range of sounds needed for the perception of words. A range between $b'-g''$ was found to be the necessary minimum, and at the most an octave below or above is used, according to Liepmann. Freund's case had this range, and *must* depend on a central lesion. Wernicke uses these data for further considerations: Our hearing covers eight octaves; only a small part is needed for the recognition of speech, and only about one fourth to one fifth of the projection fibers 'need' end in the speech center itself to make the hearing of speech possible. Hence its limitation to the posterior third or half of T_1 and the neighboring part of T_2, whereas the rest of the temporal lobe must also very largely be a terminal auditory station (on what evidence?). The possibility of a pure auditory aphasia from a double-sided lesion (Pick, etc.) limited to the entrance zone for these sounds could not be excluded, if the above reasoning concerning localization of the tone-levels were correct. A patient might indeed lose both centers for the *tone-levels* of language. But Pick's cases really were completely deaf and would seem to belong to Bleuler's pseudo-word-deafness through insufficient hearing. The right-sided path would seem to play a rôle in *restitution* since it did not take place when both sides were affected (O. Berger).

phasia), and is enacted either on request, or as repetition in the form of a question, or in states of greater general reduction, wholly automatically, as echolalia. (Bastian speaks in such cases of isolation of the auditory word-center.)

Transcortical motor aphasia is a suspension or very considerable reduction of spontaneous speech, with correct repetition and understanding of language. The utterances are not always the same words or syllables as in cortical motor aphasia, but limited to expressions of displeasure, annoyance, helplessness, and the ability to recite memory material and to repeat spoken words shows the vocabulary to be unlimited. Articulation is perfect; yet no replies except an isolated simplest answer can be obtained.

In sensory transcortical aphasia reading is done correctly, only with occasional paraphasia, but without understanding.

In motor transcortical aphasia spontaneous writing is impossible, writing to dictation correct or slightly paraphasic, reading understood, though marred by paraphasia on reading aloud.

Finally Wernicke mentions combined forms: total aphasia with loss of comprehension and utterance, usually with loss of internal language, *always with hemiplegia;* or much more rarely with fairly preserved internal language, as a summation of the two transcortical forms, at times *without* hemiplegia; further mixtures of subcortical and transcortical forms — even more frequent than the pure forms.

Reviewing briefly what Wernicke claims for the speech-function as such, we find an ' auditory word-center ' and a ' motor word-center,' and a direct and an indirect connection of the two are referred to under the common term ' word-concept ' or ' word-notion.' Partial defects (loss of only a limited number of words) are not admitted in motor aphasia. In auditory aphasia, there is no record of any dropping out of special sounds. The occasionally reported loss of special languages or dialects evidently does not command Wernicke's attention. There are several word-functions; several degrees of identification (from appreciation of the word-sound to that of the meaning), and of verbal elaboration (from recurrent utterance through automatic echolalia to paraphasic utterances and finally free spontaneous speech); but for all of these we are merely given the ' word-concept,' in one place used as that word-function which allows of decomposition of the word into letters (where the ability to write is made the criterion between cortical and subcortical aphasia), in another place as the word-function sufficient for automatic echolalia (which need not even be understood).

We now follow Wernicke to the second part of his discussion

dealing with the question whether the occasional occurrence of isolated agraphia or of isolated alexia warrants the assumption of special reading and writing centers (with Charcot and Bastian and others), or the restriction to a reading center (Dejerine), or neither (Wernicke and v. Monakow).

Written language (symbolization by written signs) is acquired late and not common property of everybody, and therefore not provided with a uniform brain-mechanism such as we assume in the whole race for symbolization by word-sound. Hieroglyphs would have a mechanism different from the method with letters, which makes of reading a process of spelling, as Wernicke maintains with Grashey and Goldscheider, with visual memories only for letters and not for words, except for a small number of very common words (especially one's name). He therefore declines the identification of a visual word picture with an object without some intermediary 'thinking' in which the letters cease to be essential and of direct meaning. Charcot's case of thinking in written words is an extreme exception not fit for generalization, as little as the hypothetical types of 'moteurs, auditifs and visuels.' Nor would it be right to generalize from deaf-mutes. Wernicke does not know visual word-memories, but only twenty-five letters and a few ready-made compounds. He does not think it likely that there should be a visual duplication of what is already available in the sound-formula. *Disorders of reading and writing* are fundamentally distinguished as *either verbal, i. e.*, depending on disorder of the word-concept, *or literal*, independent of any such disorder, but due to non-recognition of the form of the letter. Wernicke specially considers the two cases of Rieger and Sommer in which cerebral traumatism led to imperception of a limited number of letters. The patient of Grashey, who could find the words for objects in no other way but by writing and only after the entire word was written, shows according to Wernicke merely a peculiar trick, and moreover that the letters and even combinations of letters are not directly related to the object but become so only when the material for the sound-equivalent is complete. *Written language*, being merely spelled language, is *a transcortical function* subordinated to the centers of speech, dependent on its integrity, and, in return, the best criterion of the integrity of word-concepts and of internal language.

In the main, the disorders of written language (as far as they are verbal) go parallel with those of spoken language. The understanding of what is read vanishes with that of what is spoken (or at least formulated), and the ability to write spontaneously with the ability to

speak spontaneously, and paraphasia in reading aloud and in writing to dictation keeps pace with paraphasia on trying to repeat spoken words. Writing may be especially difficult because it depends not only on the ability of finding the word but of finding also the letters belonging to it.

In *cortical motor aphasia* the word-concept is, as a rule, profoundly disturbed, as shown by the *lasting alexia and agraphia* (which Bastian does not accept as due to lesion of the Broca center). The recognition and the copying of letters (even from print into writing) is, however, not involved. Yet, Thomas and Roux found that, in recovery, the patient first re-learns to read complete words, then simple syllables and, at last, single letters. Writing is apt to improve slowly, about as articulated speech, but more slowly for dictation than for spontaneous speech (Dejerine). v. Monakow errs when he minimizes the special importance of the integrity of the word concept for writing and when he claims that the motor aphasic is often able to write better than he speaks. This holds only for exceptions (Banti's case) — and v. Monakow claims for these cases disease of only the opercular lip of the Broca convolution.

Cortical sensory aphasia does not occur without very profound disorder of written language, especially agraphia is apt to be persistent, perhaps partly on account of the neighborhood of a path very essential for the motor act of writing. *Conclusive records are, however, scarce.*

The schematic presentation of the function of written language differs from the Lichtheim scheme of spoken language in the fact that evidently the motor execution cannot be roused directly from the concept-mechanisms (as the motor-speech utterance can be, without the help of the auditory center); it seems that writing always demands the rousing of the optic memory of the letters. On the other hand, the motor component is not essential for the recognition of letters, as is shown by our reading of printed letters.

The very foundation for writing is the existence of notions of direction, since we can write with any part of the body. A special center for writing movements of the right hand does not appear plausible, and is, so far, based 'on material uncritically used.'

A definite one-sided localization of the memories for letters has, however, been claimed with more appearance of justification and is upheld by Dejerine, Bastian, Pick, etc. Wernicke opposes this view with v. Monakow, as he did in his classical review of 1886, reprinted in his *Gesammelte Aufsätze.* Wernicke, to begin with, feels sure that

a visual *word*-center is not to be thought of, but at best a center for letters. Letter-images are distinguished above all other optic images by being: (1) Two dimensional and therefore 'having only one visual form,' not innumerable ones as the three dimensional objects (Storch); (2) used extremely often; and (3) devoid of a direct connection with concrete concepts, and devoid of associations apart from being connected with the one-sided speech-center, especially its auditory part. This alone does not, however, guarantee one-sided localization. Any special localization within the visual sphere is difficult to prove. There is not even a demonstration of any special cortical locality for sharpest vision, and of another locality for the most differentiated oculomotor directive concepts. The functional acquisition of letter concepts does not point solely to the left hemisphere either. Macular vision, which is almost alone concerned in the recognition of letters, is represented in either hemisphere, and large letters are equally soon recognized when approached in the right and the left visual field. Right hemianopsia may cause difficulty in reading, but it does not imply letter-blindness (even directly after the shock), although the latter is always combined with right hemianopsia. Bastian and Dejerine resort to the explanation that callosal fibers reach the specialized 'visual word-center' from the right hemisphere; but Wernicke sees in this an unjustified extension of the afferent optic path-way beyond its projection-field and a disregard of Meynert's fundamental law of the exclusively associative nature of the callosum. He claims that otherwise even the right hemianopsia would be covered up by callosal fibers from the normal hemisphere to the visual center cut off from the tract of its side (which might be relatively true if Dufour's distinction of hemianopsia with vision nulle or vision noire holds), and that the relation is quite different from that of the auditory afferent path to the auditory word-center, concerning which he says (p. 519, below) that the functional interruption of the auditory path to the left temporal lobe is the cause of the subcortical sensory aphasia, while in the only conclusive case of Liepmann he admits the importance of the participation of callosal fibers. "The facts of pathology refute the unilaterality and narrow localization of a visual word-center; what then creates the appearance to the contrary?" Evidently the close commissural relation with the one-sided speech-field, especially its auditory part, for which two possibilities are to be considered: 1. v. Monakow's view, that the focus underneath the angular gyrus cuts the afferent optic radiation of the left side, and the crossed visual-auditory commissure. The left-sided memories are not reached by stimulation. The right-sided ones

cannot be used because they cannot rouse the sound-component; reading by spelling would be lost, and reading would be limited to a few words read as a whole. Objects would be identified because their cortical representatives are connected with more than the auditory projection field ; the frequent difficulty in naming objects will be discussed on p. 276. The recognition of forms, and among them the forms of letters (identification of the same letter in different alphabets and free copying) would remain: Yet in many of these cases copying is reduced to drawing; 'and could this be the effect of a simple interruption of the crossed visuo-auditory commissure?' The sound-component alone gives the signs their sense; and, with its loss, the sense is lost.

Alexia would therefore be a mixture of a left-sided subcortical lesion and the cutting off of the visuo-auditory commissure of the right visual center, whereas the preserved left visuo-auditory connection would remain sufficient for writing.

2. Dejerine's view assumes the principle of economy also to hold in a one-sided presentation of letters, in the left angular gyrus which alone would have a connection with the auditory speech-field.

However the future will decide this dilemma (after a reliable definition will be found for what constitutes the angular gyrus!), the denial of a unilateral letter-center is necessary to formulate the problem of inquiry of the callosal radiation. It would seem that the assumption of a visual word-center would make it easy to explain the agraphia in the case reported at the outset. A lesion just beyond this center, cutting the fibers to both arm-centers, would explain it. But why should the patient have lost at the same time the ability *to draw* the simplest figure?

Redich has found 27 cases of simple word-blindness (literal blindness?) or subcortical alexia. Wernicke adds the case of a man of 62, intelligent, without speech-disorder, who also writes quite well, but who cannot read anything, neither letters nor words, nor numbers (the latter are exempt in some cases of alexia). The patient sees, and is able to copy, letters and drawings, and even then does not understand the letters, while some patients of this type actually gain an understanding by going through the motion of writing. There is right hemianopsia. The patient recognizes objects, but occasionally has some difficulty about finding the right name (without a similar difficulty on palpation?), although he recognizes it at once from among a number of names mentioned to him. (In one of Wernicke's earlier observations a similar patient could not name any objects and

had also difficulty about finding names of concrete things in spontaneous speech — evidence of a true aphasic disorder.) At first the patient had even some difficulty in correctly *recognizing* objects seen — evidence of mind-blindness as a remote symptom of the focus implying the lasting alexia — and also a similar difficulty about recognizing objects merely palpated. Mind-blindness usually implies alexia; isolated alexia is, however, usually not complicated by additional mind-blindness. The lesion in the case is probably embolic; hemiplegic symptoms disappeared again in two to three weeks; but then the patient was found unable to read the paper.[1]

Isolated simple alexia would depend on a deep seated lesion beneath the angular gyrus, with destruction of a subcortical (and a transcortical?) path and integrity of a transcortical one, passing nearer the cortex of the angular gyrus. Dejerine saw indeed an extension from the deep lesion beneath the angular gyrus (with simple alexia) extend to the cortex and to alexia + agraphia.

Rieger's patient, a sculptor of 32, developed, six months after a fracture of the skull, loss of *p*, *x* and *y* from the small German alphabet, these and *d*, *h*, *k* and *v* from the small Latin alphabet, and 14 capital letters from both alphabets: the above with the exception of *D*, and *B*, *E*, *F*, *M*, *N*, *R*, *T* and *W*. He could neither write nor identify these; also no numbers besides 0, 1, 2 and 3. He could use the available letters on dictation and copying only, and what he read was without understanding. Otherwise there was an occasional difficulty in finding a noun in spontaneous speech; he also found it difficult to name objects on vision and palpation, but always succeeded after a long while; for letters it took him about half as much time as for objects, but for the above letters there was complete abolition. A defect of retentive memory in all sensory domains was not less marked than in Grashey's case. The ability to draw was also gone; also the recitation of series. With all this there was no reduction of intelligence in a practical sense.

Sommer's case had a similar partial alexia and agraphia after an apoplexy. For several other letters there was a variable difficulty. Moreover, he could not compound even the preserved letters into words. Writing was practically abolished, but the writing of single letters was in many respects better preserved than the reading. The patient was hemiplegic for two weeks, had right hemianopsia, but,

[1] Dyslexia (Berlin) is probably akin to alexia, but merely a great fatiguability of the reading-capacity leading to Leseschеu (Bruns), and due to atheroma or syphilitic vascular disease. Hemianopsia is not a *condition*, as in alexia.

apart from the alexia and agraphia and a casual difficulty in finding a word, he was perfectly normal.

The loss implicates not only the rare letters. The constancy of the defect 'seems to exclude purely functional factors,' — we should rather say stamps the cases as quite exceptional, with but one further analogy in the literature of cerebral pathology, a case of sensory-motor aphasia with only partial but constant vocabulary, following a psychosis (Heilbronner, *Z. f. Psych. & Phys. d. Sinnesorgane*, XXIV., p. 83). Rieger himself mentions a case of hysteria with loss of the letter *H*, and keeps aloof of localizing conjectures, whereas Wernicke suggests a lesion of paths belonging to the path *ca*, from the 'word-notion' to the visual memories of letters.

Pure isolated agraphia seems to be present in the case reported at the outset. But the internal language is not intact, and the original disorder appears to have been a transcortical motor aphasia. What is left of disorder of word-concepts does not wholly explain the strikingly motor character of the symptoms. The patient has her visual memories of letters but cannot transfer them to the motor apparatus, and since these memories are bilaterally located, there should be a *bilateral* interruption of the path between receptive and emissive centers for letters and drawing. The temporary presence of left-sided symptoms might indeed speak for a bilateral lesion; but other cases seem to have depended on left-sided lesions only; some diffuse damage may, however, have suspended the function not only of the affected but also of the opposite side. Evidence of such diffuse damage would lie in the slightly indicated transcortical disorder of *speech*. Pitres' case had at first general agraphia, but after a while it was limited to the right hand (which had otherwise recovered motility); the right hemiplegia with its profound disturbances had disappeared, leaving behind right hemianopsia, with good visual acuity. The remaining agraphia of the right hand should hardly be called a 'pure (motor) agraphia'; but it is a distinctly one-sided disorder of writing, such as, in *Liepmann's* case, was simply *part of* the right-sided apraxia (the patient wrote in mirror-writing with the left hand). The permanent absence of mirror-writing in Wernicke's patient favors to his mind the possibility of bilateral lesions.

The rare cases of *isolated literal agraphia* would seem to be analogous to conduction-aphasia; all forms of agraphia in which letters can still be shaped are, however, *verbal agraphia*, a consequence of disorders of spoken language, or of connection with the 'word-concepts.' Since writing is an additional task, it may occur that sen-

sory aphasia may recover just far enough to leave out this most difficult reaction, the translation of the word into letters. This is in harmony with the observation that paraphasic disturbances are usually exaggerated in writing, or may persist in writing when they have disappeared in spontaneous speech.

True paragraphia with disfigurement of the individual letters occurs oftenest in general paralysis or other diffuse loss of memory of the forms of letters.

It is easy to understand that Wernicke brings the problem of 'Wortfindung,' *i. e.*, finding the word or naming, in close connection with reading, the finding of the word or sound for letters. The naming of letters, like that of unisensual visual perception, such as colors, took only about one half the time taken by other objects, and in Grashey's case, letters could be named at once, even without the motor help of writing. The path for naming letters would probably be the inferior longitudinal fasciculus for the left and the crossed forceps-tapetum tract for the right hemisphere. To incriminate the same path in Freund's optic aphasia, is probably incorrect. The naming of an object presupposes its secondary identification or recognition, *i. e.*, the association of the optic memory at least with the corresponding tactile memory, which is not necessary with letters. It is certainly necessary to test the naming for all sense-qualities, and to consider whether a concept does not anyhow depend largely on one sense (thunder, waltz on the auditory, and wind, warm, cold on the tactile sense). In Grashey-Wolff's case, the visual projection field seems to have been relatively best preserved; this might perhaps explain the unique fact that he found his words by the way of the letter-compounds.

In the cases of Rieger and Sommer the concepts for certain letters are lost. This has probably nothing to do with the fact that occasionally a patient can read, *i. e.*, name words, but not single letters. One of Bastian's cases could not name a solitary letter, and misread on account of marked paraphasia, but understood what he read. This may in part be an exaggeration of the difficulty caused by unaccustomed attention to a detail act, and in part to the facilitation by secondary identification (reading manuscripts from sense).

Lately Pitres has yielded to a practical need of recognizing a provisional picture of amnestic aphasia (without disorder of understanding, reading and writing). This heterogeneous group would include most cases of isolated word-blindness, and the cases of Grashey and Rieger (which are not explained simply by their defect of Merk-

fähigkeit), and a large number of cases in which the 'amnestic aphasia' is merely a residual of various disorders. In eight of ten cases the inferior parietal lobule was affected similarly to the lesion of predilection of Naunyn's 'indefinite aphasias'; lesion of the Broca convolution is probably least represented in such difficulty of naming. Amnestic aphasia consists solely in a lack of connection between concept and word; it has no definite localization and may be simulated by diffuse memory disorder. It is essential that cases with additional paraphasia should be distinguished from cases with mere difficulty in finding a word, such as a noun designating an object. What may be normal with rare words or words of a foreign language marks a symptom of defect when it occurs in the mother-tongue. In the systematic aphasia of polyglots, a stage of amnestic aphasia is apt to precede restitution. The difficulty of finding words is a special form of transcortical motor aphasia. Its climax may be reached in the actual loss of concepts as in Rieger's and Heilbronner's case, and in other cases there is at least a relative retardation of the rising of the concepts. Only the concept as a whole, not the individual sense-memory, is capable of rousing the word, with the exception of unisensual concepts. It is obvious that difficulties in the sensory spheres are most apt to so diminish the efflux to the concept mechanism as to leave it relatively inefficient in rousing the names. For the tracing of such defects Rieger's scheme is recommended.

Lack of space forces me to put off to another occasion the review of the anatomical considerations of Wernicke. What has been rendered of his general discussion cannot fail to rouse a wholesome desire for convincing observations of patients sufficiently capable of introspection to give more directness to the discussions, and with such anatomical examination as will put an end to the regretable tendency of so many clinicians to consider the white matter of the hemispheres the cornucopia of all the desirable conduction paths.

The great advances in the studies of asymbolia and apraxia will do their share in shaping new problems for the elaboration of sensory impressions into speech- and writing-reactions, and in this connection Storch's work promises fair to do away with much of the brain-cell mythology with which the theory of aphasia is afflicted, and also the hazy dogmatism about the relation of concept and word.

PSYCHOLOGICAL LITERATURE.

MOTOR PATHOLOGY.

Ueber einige seltene Zustandsbilder bei progressiver Paralyse. Apraxie, transkortikale sensorische Aphasie, subkortikale sensorische Aphasie, sensorisch-motorische Asymbolie. KARL ABRAHAM. Allg. Zeitschrift f. Psychiatrie, 1904, LXI., 502–523.

Studien über motorische Apraxie und ihr nahestehende Erscheinungen; ihre Bedeutung in der Symptomatologie psychopathischer Symptomenkomplexe. ARNOLD PICK. Leipzig, Deuticke, 1905.

Der weitere Krankheitsverlauf bei dem einseitig Apraktischen und der Gehirnbefund auf Grund von Serienschnitten. H. LIEPMANN. Monatsschrift f. Psychiatrie u. Neurologie, April, 1905, XVII. (4), 289–311.

Ueber Störungen des Handelns bei Gehirnkranken. H. LIEPMANN. Berlin, S. Karger, 1905.

As was to be expected, the studies of Liepmann and Pick have become the stimulus to analyze the great mass of inadequate reactions in mental disease, for the occurrence of symptoms which had been successfully referred to definite cerebral mechanisms.

The papers enumerated, and also one of Marcuse[1] and others, have found a very lucid systematic analysis by Professor Liepmann in the little book which has just appeared. Before mentioning its contents I may be permitted to add to the review in the PSYCHOLOGICAL BULLETIN, Vol. I., pp. 277–285, a supplementary statement from the full report of the examination of the brain of the case of one-sided apraxia. The first report did not accentuate fully enough what now seems to have been the fundamental point in the determination of that remarkably lucid case, namely, the finding of the degeneration of the corpus callosum with the exception of the splenium, so that with practical integrity of both left central convolutions there was a sequestration from the cortex of the frontal lobe by a subcortical frontal focus, and another one by a subcortical focus in the parietal lobe from both the occipital and temporal lobes.

The disorders which Abraham, Pick, Marcuse and Liepmann de-

[1] *Centralbl. f. Nervenheilk. u. Psych.*, 1904, No. 179.

scribe, and which are the topic of Liepmann's book, are briefly illustrated by the following types:

The patient of Pick is asked to light a candle; he merely approaches the burning match to the candle, but allows it to burn down and finally blows it out; that is, he begins correctly, but the aim concept dwindles down before completion of the act. Another patient of Pick's raises a pistol to his eye like a gun, although he names it correctly as a revolver; or a patient takes a tooth-brush and brushes his moustache instead of his teeth; the aim concept is side-tracked into a similar field (analogous to indirect associations); or the patient takes a cigar and match-box, opens the box and squeezes in the cigar, then rubs the cigar on the side of the match-box; that is, he mixes up the individual components of the complicated complex of activity; or the patient makes the movements of smoking with his mouth, while he has the cigar several centimeters from his mouth — omission of intermediate steps. Another patient is given a shoe-brush, but brushes an excoriated part of his hand; that is, he becomes side-tracked by an intercurrent impression; or the patient who names correctly the box with blacking is given a shoe to polish it; he grasps it, bows and rubs the shoe against his slipper. Also instances of perseveration of activity are given. The patient who has just blown out a match blows at a revolver and a cigar which are handed to him. With these reactions Liepmann compares the peculiar reactions of his cases of true motor apraxia (see last year's review), and after a keen analysis of the process of activity and the various issues under discussion he gives the following final review:

"A number of superimposed levels of the central nervous system coöperate in our activity. Certain regulations are altogether attended to by the posterior columns (with the reflex collaterals in the spinal cord) and the cerebellum without any participation of our consciousness. The connections of the sensomotorium with an intact subcortical apparatus of afferent and efferent paths makes possible the complete coördination and the prompt use of certain synergias. The sensomotorium possesses, moreover, a memory for acquired complexes or series, superimposed upon the synergias. Without the coöperation of the subcortical apparatus (for instance the afferent side of the spinal cord), but still in connection with the brain, it can direct promptly the chief agonists only. The directives of complex activities of life according to appropriate purposes reach the sensomotorium from the entire brain. Which, then, are the various ways in which activity can be damaged by brain disease?"

So far disorders of activity due to focal lesions were traced as

1. Paralysis or paresis; that is, reduction or diminution of motility.

2. Ataxia: faulty appreciation of the force of the excursion, due to defects of the peripheral kinæsthetic directives.

3. Loss of kinæsthetic conception: a picture difficult to differentiate from cortical ataxia, usually called mind-blindness.

4. Agnosia (sensory asymbolia or apraxia in the older sense): loss of recognition notwithstanding preservation of sensation. The identification of new impressions with memories fails to come about owing to the loss of the latter (Wernicke), or owing to an obstacle in the connection between the two (Lissauer).

5. An even more indirect cause of disordered activity is the dropping out of certain qualities and spatial features of sensation: Cortical blindness, hemianopsia, cortical deafness, etc. The corresponding loss for the kinæsthetic field is already described sub. 2.

We now have to insert between 1, 2, 3 on the one hand, and 4, 5 on the other, a sixth type, motor apraxia or apraxia of innervation. The movement is not in harmony with the ideatory process, the cortico-muscular apparatus works, but not in the service of the entire psychic process.

And further — 7. Ideatory apraxia, not a true focal symptom, but determined by diffuse processes, or a remote symptom of larger foci: the movements are in harmony with the ideatory process, but the latter is disturbed in that portion which serves to transform the sketch of the series of movements composing the principal aim concept into subconcepts. The ideatory apraxia probably is a part of a general disorder of ideation (memory, attention, etc.), and usually accompanies agnosia which again may be merely ideatory.

We therefore have in the chain from stimulus to movement the following causes of disordered activity in brain disease:

1. Loss of optic or auditory or tactile sensation (cortical blindness, cortical deafness, cortical anæsthesia).

2. Loss of kinæsthetic sensations and corresponding centripetal stimuli which do not come to consciousness: ataxia.

3. Agnosia — among which ideatory agnosia.

4. Ideatory apraxia.

5. Motor apraxia.

6. Loss of kinæsthetic concepts = mind palsy.

7. Paralysis or paresis.

Perseveration would enter either sub. 4, or as 8, as an independent source of disorder of activity.

Liepmann's chief point is the differentiation of ideatory and motor apraxia. Most of the instances of 'motor apraxia' mentioned from Pick in the beginning of this review, Liepmann has to refer to the group of ideatory apraxia — that is, they are disorders in which the fault lies with the proper planning and execution of the aim concept in all its details, whereas the movements themselves are really carried out correctly in harmony with the defective results of the ideatory part of the reaction. 'Motor apraxia' should be limited to disorders in which the action itself is side-tracked beyond the ideatory plan — that is, altogether in the innervatory and probably extrapsychical part of the whole biological reaction chain. For the differentiation of the two he gives the following points:

1. Motor apraxia is a disorder of individual limbs, single or in combination. In his famous case the limbs of the right side were affected; in another patient both arms, but the left one more, whereas the legs and muscles of the face were free. In Herzog's case, the muscles of the face, the left leg and left hand were especially affected; and since the right hand was not quite eupractic there may have been a certain general ideatory apraxia besides the motor apraxia. In Pick's cases he would at best see mixtures of motor apraxia, the bulk of the symptoms being ideatory apraxia.

True motor apraxia is certain where only one or a few limbs show faulty reactions, while the patient still can demonstrate the correctness of his idea of the reaction plan by adequate responses with *other* limbs. Whereas we deal most probably with ideatory apraxia where it does not make any essential difference which limb is used.

2. Motor apraxia is betrayed even in our simple acts. His patient could not even show the tongue, make a fist, extend the index or draw a simple line with his right hand.

3. Even imitation is interfered with in these simple activities (an important counter-test not to be neglected). Any attention or memory disorders which would interfere by themselves with such simple imitations would wipe out the very possibility of demonstrating motor apraxia. In ideatory apraxia the perseveration would be the only excuse for disability in such simple acts.

4. Most ideatory mistakes of activity are open to a psychological explanation; whereas motor apraxia proper points more to physical obstacles and a wholly senseless *quid pro quo* (gnashing the teeth before pronouncing a vowel, raising an ink well instead of showing the tongue); the latter instance might of course also occur through distraction in ideatory apraxia. *Amorphous movements* are most

typical, and compare with jargon aphasia. The raising of the ink well for showing the tongue in his case of apraxia might perhaps figure as secondary ideatory apraxia implanted on motor apraxia. The tests might be formulated as follows: If the motor apraxic could speak he would be able to describe his plan and intention perfectly, but explain that even with the greatest effort the afflicted limb would not carry out the plan; the ideatory apraxic, however, would, with sufficient preservation of introspection, be able to say that his arm carries out the plan, but that there are flaws in the plan.

Considering the immense variety of faulty reactions with which we deal in psychiatry, this differentiation is a valuable addition to the as yet scanty means of drawing apparently confused observations into formulas of disorders of distinct mechanisms. While we should not expect that we ever shall be able to get along without psychological descriptions and references to the relatively vaguer concepts of attention, association, etc., and while we have, therefore, no right to belittle the latter and to try to discard them, and still less to use them carelessly, a gain of definition such as Liepmann's work brings us is greatly to be welcomed. It is doing for activities what the studies of aphasia do for language, that is, for a special type of sensori-motor reaction; and as soon as such concrete analyses will be able to more and more replace the very largely imaginative hypothetical constructions of neurological psychology, we may see that neurologists will pay more attention to the actual psychological and introspective analyses of their cases.

A glance at the literature on aphasia shows that a great number of neurologists take for granted schemes of analyses and correlations with brain lesions which go much further than the actual evidence at hand justifies. This unfortunately has gone so far that many physicians believe that the problems of aphasia are about settled, and that it would almost be a discredit and a mere duplication of work to publish new observations. I cannot help but think that there will be a rejuvenation of the interest, a tearing down of many insufficiently supported assumptions; and a great deal of inspiration from the careful work of Liepmann, Pick and others in the domain of pathology of action will again arouse the hunger for similarly consciencious work in aphasia. How much improvement is needed on the anatomical side is effectually shown by Flechsig, whose recent article is considered in the final review (p. 288). A. M.

Klinische und kritische Beiträge zur Lehre von den Sprachstörungen. GUSTAV WOLFF. Leipzig, Veit & Co., 1904.

As a critical study for any one working on aphasia this little book is strongly to be recommended. It shows very forcibly how the reasoning from a schematic plan of the function of speech has led many investigators to the as yet unwarranted belief that there was such a thing as optic aphasia, dependent on an interruption of the path connecting the visual perception center and the speech center, leading to an inability to name objects seen, although they are recognized. Wolff shows very clearly that in all cases recorded so far, the inability to name objects seen was either due to an inability to recognize the object, or part of a general speech disorder, which referred to the utilization of tactile or other sensory impressions as well, or that the isolated inability occurred only with objects in which the optic impression is subordinate, so that the tactile appreciation was anyhow more likely to be needed for naming. He shows great haziness among the advocates of optic aphasia as to whether the connection of the optic perception center should be one with the auditory or with the motor speech center. Wolff discusses the cases of thirteen other authors and compares them with three cases of his own.

The first one of Wolff's own cases, a man of fifty-nine, practically blind with retinitis pigmentosa, had a slight shock with transitory paresis of the right side, and when two days later he spoke again, he was unable to name any objects felt or heard, although he recognized and used them correctly. The only exception were parts of his body, at least when they were pinched, but not those of another person. Examination of taste and smell were neglected. The patient succumbed to a renewed attack a fortnight later. A subdural blood clot of the size of a fist pressed upon the left parieto-temporal cortex. Wolff mentions this case as an instance of isolated integrity of one set of names, without adequate explanation, but hardly any justification of a special localization of these names.

The second case is one of simple demented general paralysis in a woman of fifty-six, who lost the ability to name objects although she recognized them. The various senses behaved in the same way; but the names of the parts of the body again formed an exception. There was, however, also a great reduction of spontaneous speech while repetition was preserved. The anomia was therefore part of a 'transcortical motor aphasia.' In harmony with most cases of this type, there was merely a diffuse lesion.

The third case, a woman of sixty-six, with senile confusion, came

out of an apoplectic attack with lasting speech defect: great restriction of spontaneous speech to a few simple requests, with preservation of most numbers. She understands, but is unable to name any object, no matter through which sense, although she shows recognition by correct use of the same. The parts of the body are included in this anomia, also articles of food which she eats, although she points correctly to things named to her. Reading and writing abolished; repetition of words perfect. Death in a renewed apoplexy. Besides a fresh hæmorrhage destroying the right basal ganglia, there was a cyst of nearly walnut size in T_3, just in front of the occipitotemporal notch. The picture was again a transcortical motor aphasia; superficial examination might have led to the diagnosis of optic aphasia, but it was also tactile, acustic and gustatory. Anatomically the case is an exact corroboration of the finding of Mills. Wolff does not, however, speak of a naming center, but admits the relative frequency of this symptom 'anomia' in abscess formation in this region.

In view of the haziness of the center concept, and of the difficulty of determining what is to be referred to lesion of the cortex and what to the numerous underlying fiber-paths of this region, it would indeed be a mistake to claim more than that lesion of this area is apt to lead to anomia, but that anomia may also be a symptom of diffuse lesions.

A. M.

MULTIPLE PERSONALITY.

Multiple Personality. An Experimental Investigation into the Nature of Human Individuality. Boris Sidis and Simon Goodhart. New York, Appleton, 1905. Pp. 462.

The present contribution of Dr. Sidis, in association with Dr. Goodhart, to the comprehension of personality in its abnormal manifestations centers about the remarkable case of Mr. Hanna; and for this alone the volume at once assumes an important place in the literature of this perplexing topic. The case is noteworthy in many aspects; it is minutely and ably reported; the patient is a man of unusual intelligence and education, as well as a person of normal good health; and the appearance of the altered personality comes suddenly by reason of an accident, while the acquisitions of the entire experience up to the moment of the accident disappear. In contrast with the usual cases in which alterations of personality are developed slowly in hysterical patients, the new order of things following upon more or less protracted periods of psychic incubation, and merging in puzzling ways factors of the old personality with the new development, Mr. Hanna's case is that of the most complete loss of the

personal acquisitions and the memory thereof that has yet been recorded. It is thus in a very true sense the psychologist's case of altered personality, for it corresponds most nearly to the conditions which the psychologist would choose, were he able to experiment in this field; and in the end under Dr. Sidis' skillful management the actual experiment is performed, and successfully, of reinstating the original personality, so that at the present moment Mr. Hanna is substantially the same as before his curious experience. It is difficult to summarize the case itself; but it may be stated that, as the result of a fall, Mr. Hanna found himself practically as a new-born babe, with no language, no comprehension of the meaning of things, no memory-images of what sensations were or how they were to be interpreted, no knowledge of his family, or of his surroundings, or of any of the innumerable factors which constitute experience. He actually had to discover the use of his muscles and of his senses, to be taught the simplest rudiments of that practical education which occupies infancy, and yet went through all this with something of the adult facility, and, as proved later, with underlying remnants of his former adult consciousness. That his acquisitions were gained at an extremely rapid rate the story emphasizes; and the happy ending shows that at no time were the older experiences really destroyed. They had been merely suddenly and mysteriously estranged from voluntary recall, but remained in subconscious possession. The first distinct evidence of this reaction was obtained from such accounts as the patient could give of his dreams. These fell into two varieties; the one concerned with the events and modes of response of his new child-like personality, while the other, which he spoke of as vivid dreams, were traceable to real happenings of his former self. Following this suggestion, the patient was taken to New York city and there subjected to the violent assailing of his senses by means of the complex stimulations of the metropolis, in the hopes that as such experiences were not unfamiliar to the former self, they would serve by their very intensity and complexity to break through the shallow crust that at this juncture separated the conscious from the subconscious acquisitions, and thus to reinstate, even though intermittently, the older life. This actually occurred on awakening from a sound sleep during the first night following these experiences. The patient awoke as the original Mr. Hanna, with much astonishment found himself in strange surroundings, with unfamilar companions, and at once demanded the sequence of events from the moment of the accident several months before. The new state did not last long; the patient became drowsy, and

awoke in the morning with no knowledge of the night's events, though quite clear as to the experiences of the evening before. Gradually these reinstatements became more frequent and of longer duration, and resulted in an intense and painful struggle which Mr. Hanna afterward recounted as one of the most trying moments of his life, when he really seemed forced to choose between the two personalities, each of which seemed to claim him as its own, and yet with no exclusive right. The saving alternative, which was the issue of the struggle, was to embrace them both, to merge the two, though with imperfect conviction, until they gave way to the normal state of affairs.

The case is important and interesting, not only by reason of its general progress, but on account of the many detailed observations that enrich the account, and suggest at each step well-formulated and specific problems in regard to psychological principles and analyses. These are further discussed in a series of introductory chapters, and in another series of concluding chapters, in which Dr. Sidis presents his general statements in regard to the nature of personality. The trend of these is not easily reproduced, and indeed leaves upon the reader something of a vagueness of impression that is inevitable in our present imperfect understanding of these cases. What is more important is that the interpretation, so far as it goes, is intimately allied with the sanest and safest interpretations of modern psychology, and emphasizes the fundamental importance of the normal subconscious life as the proper starting-point for the interpretation of the abnormal. For all of these merits the volume deserves, as it doubtless will find, a useful place in the psychologist's equipment for the comprehension of the varieties and the variations of personality.

J. J.

NEGATIVE SUGGESTIBILITY.

Negative Suggestibility, a physiological prototype of negativism, of contrary auto-suggestion and certain obsessions. PROFESSOR BLEULER. Psychiat. Wochenschrift, Nos. 27 and 28, 1904.

The finest movements are obtained by combination of antagonists and agonists, representing the excess of the power of the agonists. All the peripheral mechanisms such as the heart, intestines, vessels and sphincters have their stimulant and inhibitory nerves. In psychic activity, too, any topic of thought inhibits all the other noncorrelated concepts. If, after all, thought does not always move in one direction, it would seem that association is not merely a selection of favorable and positive tendencies, but that there is at the very bottom of the

mechanisms of association a provision for the response of directly opposite tendencies. This phenomenon might be classed as association by contrast, but it is so generally present that there is much to be said in favor of the view that there is a special mechanism, a general tendency to associate with every concept also its opposite.

Bleuler gives a number of instances of this principle in the ordinary play of motives in deliberation. In children he notes the balancing of reluctance and eagerness, even where timidity is excluded; he refers to the mixtures of coyness and sexual desire, the balance of fear and eagerness in risks; the tendency to continually touch a painful tooth.

In harmony with their purpose these coupled contrasts appear to arise especially in connection with action. "Those persons who entirely exhaust the pros and cons beforehand and have completely settled their deliberation before they begin to act are rare types." With most people a decision arouses new opposite concepts; but in only a small number a decision suppresses them altogether. In such cases the association of contrasts becomes decidedly undesirable, and it is especially striking that it manifests itself much less in calm deliberation than just before action, where such an elementary mechanism is a truly fundamental protector. In this respect there are profound differences of character.

Bleuler refers to analogous contrasts in feelings, such as laughing in an accident, the perverse actions of unfavorable autosuggestions where a person anticipates the possibility of a headache, or of menstruation at an undesirable date; the tendency to acquire mannerisms which one criticises and ridicules in others; the inability to sleep owing to the excessive eagerness to sleep; the peculiarity of a blocking of the very thoughts one needs (in examinations, etc.); a fear of failure does not explain sufficiently the direction of the inhibition. Everything points to a special mechanism which tends to rouse contrasting or antagonistic concepts. It is especially marked in relatively suggestible individuals as a protection against being taken by surprise; but in the abnormal, too, negativism and suggestibility, automatism and echopraxia, frequently go hand in hand (in dementia præcox), or excessive confiding and yielding beside distrust and obstinancy (in senile dements), or suggestibility beside uncontrollable contrary autosuggestion (in hysteria), etc. Wherever an emotion, or the narrowing of the field of consciousness, or a blocking process, interferes with the course of thought, the elementary process of associative contrasts becomes prominent.

Suggestibility is a certain side of affectivity; as such it is connected more closely with volition and activity than with ideation; the laboratory test leads to fewer contrast associations than actual life with its volition and activity. Timidity and misoneism are affects of negative suggestion. Emotionally important ideas are especially apt to give rise to negative suggestions when the impulsiveness of activity makes one more in need of this control. As suggestion generally, and affectivity, negative suggestion has an enormous influence also on the physical functions.

Bleuler promises perspectives for the explanation of many symptoms of dementia præcox as qualitatively and quantitatively disfigured mechanisms of normal mental life (through predominance of this fundamental appeal to contrast). He also points to many obsessions, to the contrary autosuggestions of hysteria, and finally refers to some explanations of this phenomenon by other writers.

A. M.

METHODS OF BRAIN RESEARCH.

Einige Bemerkungen über Untersuchungsmethoden der Grosshirnrinde, insbesondere des Menschen. PAUL FLECHSIG. Sitzungsberichte der Akad. Wiss. Leipzig, Math.-phys. Klasse, Sitzung vom 11. Jannar, 1904. Pp. 50–104 and pp. 177–248 (with 4 plates).

Flechsig herewith presents to the central committee of brain investigation a critical comparison of the various methods of neurological research, with special reference to his myelogenetic method. For some reason Flechsig has met with emphatic opposition at the hands of most neurologists. By making certain peremptory claims, and by an unfortunate personal vein, he brought upon himself a flood of bitter attacks, which does not reflect very pleasantly on the brotherhood of neurologists. His latest paper gives a rather fair comparative estimate of the fundamental methods of research and an outlook towards better dovetailing of the accessible methods.

In view of the fact that there is already a full review of the essentially anatomical part of the paper of Flechsig, with a copy of several illustrations, from the pen of Florence R. Sabin, in the *Johns Hopkins Hospital Bulletin*, Vol. 16, Feb., 1905, pp. 45–49, we can limit ourselves to a few remarks concerning the present status of neurological and neuro-pathological problems.

Flechsig acknowledges the need of a comparative study of all the types of nerve cells and their combinations (such as has its principal representatives in Cajal, Campbell, etc.). He recognizes that most of

our anatomical data refer to the theory of *conduction*-paths which does little for the explanation of the intrinsic activity, for which, he thinks, we may have to await a greater development of our knowledge of histo-chemistry. In the main he stands by His's conception of neurones without deciding for or against continuity *vs.* contiguity. For the study of lamination of the cortex he prefers Ehrlich's methylene blue method to the Golgi method; and for the division into histologically characteristic fields or areas, he mentions the lower monkeys as especially favorable objects. He at once passes to his division of the human cortex into myelogenetic fields based on serial sections of 56 human brains at various stages of development, and leading to a division into twelve areas medullated before birth and 24 additional areas medullated after birth. His plates cannot fail to convince one of the fact that the myelogenetic subdivisions might mark rather fundamental entities. The demonstration of a zone like the field twelve, an isolated area at the transition of T_1 and the lateral occipital gyri (gyrus subangularis Flechsig), in the midst of a large area of tardy evolution (the posterior 'association-field'), and not specially isolated by any other method so far, should suffice to serve as a vindication of the method as a pathfinder. On the other hand, the subdivisions fail to differentiate zones which we know to be quite distinct, as the anterior central and the posterior central gyri, which Flechsig's method brings out as a unit. The definition which the method gives to the visual and auditory areas, and the striking coincidence with the results of other trustworthy methods, vindicates an important position to the researches of Flechsig. His results appear to do justice to a hunger for definition of areas which the literature on coarse surface anatomy failed to satisfy as soon as it had passed the stage of blindly accepted schematic drawings, and encountered the differences of individuals and races without really establishing any decisive types of gyration. To-day, Flechsig's scheme has the advantage of being in the simple schematic period. Up to a certain stage, myelinization picks out the very foundation lines of the architecture of conduction paths. But the method soon reaches its limitation, as the history of our knowledge of the cerebral afferent path (fillet) shows. Flechsig thought it to be continuous from the nuclei of Goll and Burdach to the cortex, while we know that hardly a solitary fiber of the fillet passes beyond the thalamus. As long as a bundle of fibers shows its base in a definite cell-cluster and a free cone of growth, the method is excellent; but when the bundles become entangled, the difficulty shown by the history of the fillet is repeated, and, in the jungle of fibers of the centrum

ovale, brought to a hopeless climax. Another difficulty is that the method deals with the delineation of transitory conditions and that it is not always possible to find evidence by the same method as to when to consider the decisive stage to be completed. The hope that comparison of the maturation of *function* would help in the determination of the moment, would be precarious considering the fact that the newborn rat has *no* medullated fibers, but at least an organized sucking-reflex, and that in an encephalic monster fibers become medullated in which function was wholly out of the question. For this reason it would seem safer to encourage a mapping out of the cortex with lasting conditions as a guide, such as the distinctions in lamination and cell-types determined by Campbell (*Journ. of Mental Science*, 1904, pp. 651–659), Brodmann, Bolton and others.

The very instructive summary of Dr. Sabin could easily be shown to have derived some of its most sharply defined data from the results of methods outside of the domain of myelogenetic studies. I refer to her abstract of the description of the pyramidal tract. On p. 184 Flechsig begins with erroneous interpretations of Dejerine's Fig. 49 of Vol. II., bluntly identifying Dejerine's faisceau moyen du pied du pédoncule cérébral with the pyramidal tract. The whole discussion from p. 183 to p. 191 is full of controversies which show that it is less Flechsig's myelogenetic method alone than conscientious correlation of the results of all the methods (and especially the degeneration-method) that allows the anatomical data of Flechsig's review to be formulated as concisely as has been done by Dr. Sabin. Considering that the personal element will play an important rôle in the interpretation of the rather delicate shades of myelinization, and considering the discrepancies of the results of the degeneration-method, one might fairly dread the day when the myelogenetic method would pass from Flechsig's practical monopoly into the hands of as great a number of men as would seem to make the method of degenerations unsafe to-day, according to the verdict of Flechsig.

Flechsig's searching criticism brings out valuable methodological hints. Most of the controversy about Türk's bundle, the supposed radiation from the angular gyrus and other parts, demonstrates conclusively how essential it is that pertinent cases should be published in full, and with ample illustrations, and interpreted with much more reserve than seems to be customary. Flechsig's paper is indeed a most convincing document in favor of greater systematization of central institutes for the study of the brain, with adequate funds for publication. Over and over again he can demonstrate that illustrations

and descriptions of Dejerine and v. Monakow fail to establish the proof that certain fibers are entering cortical areas because they do not allow one to judge how deeply the actual lesions cut into tracts merely passing in the depth. Full publication only will make it possible to determine the relation of the actual lesions and the fields of Flechsig or the subdivisions of the cortex on ground of architectonic studies of the permanent differences, and give the material permanent value.

As is well known, Flechsig establishes primordial zones (the primary sensory areas and certain as yet uncorrelated 'automatic' zones), which alone are said to have projection-fibers (including the cortico-thalamic connections, etc.), and each of these zones is surrounded by a ring of marginal zones whose medullation is not merely a concentric enlargement of the primordial zones but takes place in definite fields. There remain 3 larger fields, the central areas or terminal fields of the parietal, temporal and frontal lobes.

Each primordial zone probably has its corticopetal sensory and its corticofugal motor paths (the terms sensory and motor used in an extremely vague manner!). The marginal fields have their principal connection with their primordial zone, and belong to that sense quality. The large terminal areas are, however, collaborators of more than one sense, and essentially provided with long association-paths, without any connection with the nuclei of the thalamus.

Flechsig accepts as limit of the motor area Sherrington's outline in the gorilla (the anterior central gyrus and paracentral lobule), the central sulcus forming its posterior border. The posterior central gyrus alone forms the equally sharply limited sensory zone for skin, muscles, and joints. (Lesion of the parietal cortex 'never' produces sensory disorders.) The visual area is limited to the zone with Vicq d' Azyr's stripe. The 'higher visual center of many writers' is a polyæsthetic field. Eye movements are obtained only from the zone with Vicq d' Azyr's stripe and by no means from the inferior parietal lobule (the frontal center for eye-movements seems not to belong to the motor area; it has no projection-fibers and behaves in other ways differently from a motor center — p. 217).

A brief discussion of aphasia brings out some points of fundamental importance. Flechsig identifies the auditory word center with the transverse temporal gyrus, but only for pure perceptive word-deafness without affection of the word memory. Associative word-deafness may occur with complete integrity of the left auditory sphere. In this connection he would think of the marginal fields of the auditory sphere in the insular, temporal and parietal lobes, and also of the

terminal or central fields of these lobes. The Broca convolution consists of an upper (or really posterior) third, field 18*b*, which is the marginal zone of the facial hypoglossal accessory region of the anterior central gyrus, and of a 'middle third' (field 27), the transition into the orbital part, much more strongly developed in man than in anthropoids. In the scheme of cortical mechanisms of aphasia the Broca and Wernicke points may be considered as fixed (however, their function?). Flechsig also considers necessary the assumption that the angular gyrus supplies the union of letter images and sound concepts 'largely an act of memory which can only be conceived as being attached to the gray substance.'

Wernicke and v. Monakow's attempt to explain alexia as merely a disorder of a direct association system between the visual and auditory spheres, is an accumulation of errors, since the inferior longitudinal fasciculus is part of the optic projection and has nothing to do with aphasia. Moreover, Flechsig repudiates Wernicke's assumption that the word memories or auditory images are all dependent on the auditory sphere, even if one should accept the whole temporal lobe for it, as Wernicke does without any evidence. Amnestic aphasia depends on a much more extensive zone, extending to the angular gyrus. Hence the participation of the parietal lobe in the reproduction of auditory word images. Attempts to determine all the cortical regions, lesion of which completely wipe out auditory word images, will probably force us to return to multiple localization of every concept and word notion, in probably as many fields as are participating in the associations of the word-sound. Is this a negation of a real sharply outlined speech-field?

F. evidently does not imply this, as he declares himself to be in harmony with Dejerine. He points out the inaccuracy of Monakow, who sees the auditory word center only in the posterior part of T_1. (As a matter of fact Dejerine[1] assumes that lesion of T_1 only accounts for pure word-deafness, whereas word-deafness involving also spontaneous speech would depend on lesion of the second temporal gyrus. It is perfectly obvious that even on such an elementary point an agreement is not reached, and that much more careful examinations and reports of cases are needed).

The intercortical association systems have the same principle of myelinization by fields and bundles as the cortex. Flechsig raises the question whether each individual sensory sphere is connected with all the other cortical fields, or their majority, by arcuate fibers and long

[1] *Revue Neurologique*, No. 15, 1904, p. 811.

association systems, or whether each individual sensory sphere communicates with several or all of the primary sensory spheres. He declines both questions. There is no evidence that the posterior central gyrus is directly connected with any one of the other primary sensory spheres, whereas such connections exist with most of the fields 16 to 36. There may be association systems between various parts of the olfactory and gustatory spheres, and between the various segments of the central zone, but there is no evidence of tactile-visual, visual-auditory, and olfactory-auditory connections. The intervening fields are for this reason worth being called association centers. The interpolation may be either two or several marginal zones, or, in addition, one of the terminal or association fields. It is a problem of more conscientious and minute search for loss of definite sensory memories to determine to what extent the association centers have a direct relation to the memory of external sensory impressions, impulses of innervation, etc.

Flechsig adds a brief argument with Wundt, who shares Flechsig's opposition to Munk's view that the cortex consists merely of sensory centers, but is afraid of phrenological tendencies. According to Wundt the cortical sensory sphere is not merely a central repetition of the peripheral sensory surfaces, arranged so as to make them accessible to the consciousness which resides there. Flechsig also declines this view. 'With most physiologists and physicians' he holds that the sensory spheres produce consciousness in connection with sensory impressions, or really out of them. They unify the impulses carried in by the separate elements of the peripheral nerves, preserving the specific energy which depends on the peripheral terminal apparatus. The collaboration of the various senses has anatomical mediators (association-fibers) serving in the psychological associations. The majority of the cells of the second, third and fifth cortical layer of Meynert and their fibers, serve the principle of association, and certain parts of the cortex are made up of them exclusively. This is essential for all compound concepts, and especially for the connection of percepts with words. To avoid unnecessary identification with association-psychology, Flechsig would feel inclined to concede to Wundt the term of 'higher psychic centers,' for what he calls association-centers in the anatomical sense, leaving the share of each center in the higher psychic functions to the future. Flechsig does not assign a special independent psychic function to any of the thirty-six fields, but merely assumes for each a specific *share* in the psychic life as a whole. He still favors the opposition of a posterior large association-field, and of a frontal one; but

hesitates about the opposition of a cortical field for the perception of the outside world (posterior field) and those for the ego and somatic personality (frontal field). What he said of the frontal field does not conflict with Wundt's center for apperception; and the possibility of a natural anatomical subdivision of the large posterior field may furnish firm lines worth heeding by the student of psychogenesis whose material lacks principles of equal definition.

In one sense the sensory fields are also association centers, for the impressions of the same sense; but Flechsig withdraws the suggestion that the central (perirolandic) zone might also form a connecting link between all the large association centers; he claims that the long association paths from the three large terminal fields to the Rolandic part of the central zone are in part the paths for voluntary conceptual excitations of the motor centers; in part they conduct in the other direction. That man should have no association-centers but merely enormously extended sensory spheres wholly out of proportion with the sensory nerves themselves would be 'eine Ungereimtheit.' Moreover, the limitation of the projection-fibers to the primordial sensory fields is established 'beyond doubt,' and the existence of polyæsthetic fields (the parietal—visual, auditory and tactile, and the temporal—visual, auditory, tactile, gustatory and olfactory—terminal zones or association fields) is used emphatically for his justification. For the recognition of special marginal zones, the peculiar mode of myelogenetic accession gives some helps to the general need of greater accuracy and practical perspectives.

Flechsig criticises C. and O. Vogt very severely for trying to compare the relations of cat and dog with those in man. Even in Macacus he suspects that there are only marginal zones, and no true terminal fields; the anthropoids, however, have large association-centers. He finally refers to a parallelism of the phylogenetic and the myelogenetic data.

The last few years have done a great deal to take the subdivision of the brain out of mere studies of gyration into the sphere of more widely correlated interests. The surface anatomy has gained much by various workers. Among them Flechsig certainly deserves much credit, and his subdivisions will be very useful until they are superceded by more accurate ontogenetic results. A. M.

BOOKS RECEIVED FROM JULY 5 TO AUGUST 5.

L'art de vivre. Dr. Toulouse. Paris, Bibl. Charpentier, 1905. Pp. iv + 310.

A Preliminary Report on the Protozoa of the Fresh Waters of Connecticut. Herbert Williams Conn. Hartford (Conn.), Case, Lockwood & Brainard Co.—Hartford Press, 1905. Pp. 69 + 34 plates.

Ueber den Ursachenbegriff im geltenden Strafrecht. Adolf Reinach. Leipzig, J. A. Barth, 1905. Pp. 69.

Schiller's Stellung in der Entwicklungsgeschichte des Humanismus. Ludwig Keller. Berlin, Weidmannsche Buchh., 1905. Pp. 87.

Index Philosophique — Philosophie et Sciences. 2e Année, 1903. N. Vaschide. (Publ. ann. de la Revue de Philosophie.) Paris, Chevalier & Rivière, 1905. Pp. 464. [Contains 5,367 titles with brief résumé of many of the more important.]

La psychologie peut-elle être une science explicative? Ed. Claparède. (Repr. fr. C. R. 2e Congrès Int. de Philosophie.) Pp. 4.

Esquisse d'une Théorie biologique du Sommeil. Ed. Claparède. (Repr. fr. Arch. de Psychol., IV.) Pp. 245–349.

La psychologie comparée est-elle légitime? Ed. Claparède. (Repr. fr. Arch. de Psychol., V.) Pp. 13–35.

Lo spiritismo secondo Shakespeare. N. R. D'Alfonso. Rome, Loescher, 1905. Pp. 47.

NOTES AND NEWS.

Professor Carl Wernicke, of Halle, was killed on June 15 in a bicycle accident. His death is a great loss to psychiatry. He was one of the most independent and purposeful investigators of the functions of the brain, including psychiatry. When only twenty-six years old he wrote his classical paper, 'Der aphasische Symptomencomplex' (1874). Among his pupils may be mentioned Sachs, Lissauer, Liepmann, Bonhoeffer, Heilbronner and Gaupp. It is a curious coincidence that this number of the Bulletin contains an exhaustive review and discussion of his recent important contribution on aphasia.

WILLISTON S. HOUGH, PH.D., formerly of the University of Minnesota, has been appointed professor of philosophy in the George Washington University at Washington.

DR. KUHLMANN, now of Clark University, has been appointed assistant in psychology in the University of Wisconsin.

IT is announced that Professor Josiah Royce, of Harvard University, will give a course of lectures at the Johns Hopkins University in January, 1906, on 'Aspects of Post-Kantian Idealism.'

THE following are taken from the press:

DR. ERNST MEUMANN, of Zurich, has been called to the chair of philosophy at Königsberg.

DR. H. K. WOLFE, formerly professor of philosophy at the University of Nebraska and recently principal of the Lincoln High School, has been elected professor of philosophy and education at the University of Montana.

MR. WILLIAM HARPER DAVIS, instructor in philosophy and psychology at Lehigh University, has been elected assistant professor, in charge of the department.

DR. J. W. HICKSON has been appointed assistant professor of psychology and lecturer in philosophy at McGill University.

PROFESSOR GEORGE T. LADD, who has resigned from the chair of philosophy at Yale University, has arranged to pass the latter half of next year as professor of philosophy at Western Reserve University.

THE council of the University of Liverpool has instituted a lectureship in experimental psychology. The work in psychology will, for the present, be carried on in the physiological laboratory.